```
=> fil reg; d ide l3 1-2; d ide l12
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DICTIONARY FILE UPDATES:   2 MAY 2007  HIGHEST RN 934214-84-3

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TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

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http://www.cas.org/support/stngen/stndoc/properties.html


L3   ANSWER 1 OF 2  REGISTRY  COPYRIGHT 2007 ACS on STN
RN   37076-69-0  REGISTRY
ED   Entered STN:  16 Nov 1984
CN   9H-Purin-6-amine, 9-[(2R)-tetrahydro-2-furanyl]- (9CI)   (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   9H-Purin-6-amine, 9-(tetrahydro-2-furanyl)-, (R)-
FS   STEREOSEARCH
MF   C9 H11 N5 O
LC   STN Files:   BEILSTEIN*, CA, CAPLUS
        (*File contains numerically searchable property data)

Absolute stereochemistry.
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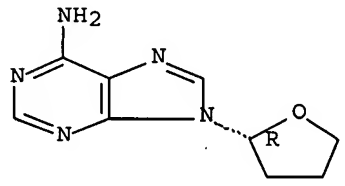


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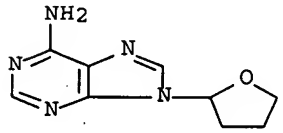
**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

               3 REFERENCES IN FILE CA (1907 TO DATE)
               3 REFERENCES IN FILE CAPLUS (1907 TO DATE)

L3   ANSWER 2 OF 2  REGISTRY  COPYRIGHT 2007 ACS on STN
RN   17318-31-9  REGISTRY
```

ED Entered STN: 16 Nov 1984
CN **9H-Purin-6-amine, 9-(tetrahydro-2-furanyl)-** (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Adenine, 9-(tetrahydro-2-furyl)- (7CI, 8CI)
OTHER NAMES:
CN 9-(Tetrahydro-2-furyl)adenine
CN NSC 53339
CN SQ 22536
CN Tetrahydrofuryl-9-adenine
DR 115016-69-8
MF C9 H11 N5 O
LC STN Files: AGRICOLA, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, DDFU, DRUGU, EMBASE, MEDLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

60 REFERENCES IN FILE CA (1907 TO DATE)
61 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L12 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
RN 9012-42-4 REGISTRY
ED Entered STN: 16 Nov 1984
CN Cyclase, adenylate (CA INDEX NAME)
OTHER NAMES:
CN Adenyl cyclase
CN Adenylate cyclase
CN **Adenylyl cyclase**
CN E.C. 4.6.1.1
MF Unspecified
CI MAN
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BIOSIS, BIOTECHNO, CA, CABA, CAPLUS, CASREACT, CBNB, CIN, CSCHEM, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, PROMT, TOXCENTER, USPAT2, USPATFULL

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

21797 REFERENCES IN FILE CA (1907 TO DATE)
57 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
21811 REFERENCES IN FILE CAPLUS (1907 TO DATE)

INVENTOR SEARCH

=> => fil biosis; d que l34; fil biotechno; d que l98; fil capl; d que l10; fil drugu; d que l84; fil embase; d que l61; fil medl; d que l48
FILE 'BIOSIS' ENTERED AT 16:02:34 ON 03 MAY 2007

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 2 May 2007 (20070502/ED)

L27	434	SEA FILE=BIOSIS ABB=ON	JANG I?/AU
L28	119	SEA FILE=BIOSIS ABB=ON	YEO E?/AU
L29	10351	SEA FILE=BIOSIS ABB=ON	PARK S?/AU
L34	4	SEA FILE=BIOSIS ABB=ON	(L27 AND L28 AND L29)

FILE 'BIOTECHNO' ENTERED AT 16:02:34 ON 03 MAY 2007

FILE LAST UPDATED: 7 JAN 2004 <20040107/UP>
FILE COVERS 1980 TO 2003.

>>> BIOTECHNO IS NO LONGER BEING UPDATED AS OF 2004 <<<

>>> SIMULTANEOUS LEFT AND RIGHT TRUNCATION AVAILABLE IN /CT AND BASIC INDEX <<<

L3	2	SEA FILE=REGISTRY ABB=ON	("9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-FURANYL)-"/CN OR "9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-FURANYL)-, (R)-"/CN)
L90	59	SEA FILE=BIOTECHNO ABB=ON	JANG I?/AU
L91	22	SEA FILE=BIOTECHNO ABB=ON	YEO E?/AU
L92	1417	SEA FILE=BIOTECHNO ABB=ON	PARK S?/AU
L93	78	SEA FILE=BIOTECHNO ABB=ON	L3
L94	84	SEA FILE=BIOTECHNO ABB=ON	(((TETRAHYDRO OR TETRA HYDRO)(1W)FURYL) OR TETRAHYDROFURYL)(1A)ADENINE
L97	75	SEA FILE=BIOTECHNO ABB=ON	NSC53339 OR NSC 53339 OR SQ22536 OR SQ 22536
L98	2	SEA FILE=BIOTECHNO ABB=ON	(L90 AND L91 AND L92) OR ((L90 OR L91 OR L92) AND (L93 OR L94 OR L97))

FILE 'CAPLUS' ENTERED AT 16:02:35 ON 03 MAY 2007

FILE COVERS 1907 - 3 May 2007 VOL 146 ISS 19
FILE LAST UPDATED: 2 May 2007 (20070502/ED)

Effective October 17, 2005, revised CAS Information Use Policies apply.
They are available for your review at:

http://www.cas.org/infopolicy.html
'OBI' IS DEFAULT SEARCH FIELD FOR 'CAPLUS' FILE

```
L1             1 SEA FILE=CAPLUS ABB=ON  US2005-517269/AP
L3             2 SEA FILE=REGISTRY ABB=ON  ("9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-F
                 URANYL)-"/CN OR "9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-FURANYL)-,
                 (R)-"/CN)
L4            64 SEA FILE=CAPLUS ABB=ON  L3
L5           529 SEA FILE=CAPLUS ABB=ON  JANG I?/AU
L6            80 SEA FILE=CAPLUS ABB=ON  YEO E?/AU
L7         22862 SEA FILE=CAPLUS ABB=ON  PARK S?/AU
L8             7 SEA FILE=CAPLUS ABB=ON  L5 AND L6 AND L7
L9             2 SEA FILE=CAPLUS ABB=ON  (L5 OR L6 OR L7) AND L4
L10            8 SEA FILE=CAPLUS ABB=ON  (L1 OR L8 OR L9)
```

```
FILE 'DRUGU' ENTERED AT 16:02:35 ON 03 MAY 2007
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FILE LAST UPDATED: 3 MAY 2007       <20070503/UP>
>>>  DERWENT DRUG FILE (SUBSCRIBER)  <<<

>>>  FILE COVERS 1983 TO DATE  <<<
>>>  THESAURUS AVAILABLE IN /CT  <<<
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```
L3             2 SEA FILE=REGISTRY ABB=ON  ("9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-F
                 URANYL)-"/CN OR "9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-FURANYL)-,
                 (R)-"/CN)
L76          113 SEA FILE=DRUGU ABB=ON  JANG I?/AU
L77           18 SEA FILE=DRUGU ABB=ON  YEO E?/AU
L78          778 SEA FILE=DRUGU ABB=ON  PARK S?/AU
L79            7 SEA FILE=DRUGU ABB=ON  L3
L80           76 SEA FILE=DRUGU ABB=ON  SQ-22536/CT
L82            0 SEA FILE=DRUGU ABB=ON  (L76 OR L77 OR L78) AND (L79 OR L80)
L83            0 SEA FILE=DRUGU ABB=ON  L76 AND L77 AND L78
L84            0 SEA FILE=DRUGU ABB=ON  (L82 OR L83)
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```
FILE 'EMBASE' ENTERED AT 16:02:36 ON 03 MAY 2007
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 FILE COVERS 1974 TO 3 May 2007 (20070503/ED)
```

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L53          294 SEA FILE=EMBASE ABB=ON  JANG I?/AU
L54           79 SEA FILE=EMBASE ABB=ON  YEO E?/AU
L55         6263 SEA FILE=EMBASE ABB=ON  PARK S?/AU
L61            5 SEA FILE=EMBASE ABB=ON  L53 AND L54 AND L55
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FILE 'MEDLINE' ENTERED AT 16:02:36 ON 03 MAY 2007

FILE LAST UPDATED: 2 May 2007 (20070502/UP). FILE COVERS 1950 TO DATE.

This file contains CAS Registry Numbers for easy and accurate substance identification.

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L45          282 SEA FILE=MEDLINE ABB=ON  JANG I?/AU
L46           89 SEA FILE=MEDLINE ABB=ON  YEO E?/AU
L47         7065 SEA FILE=MEDLINE ABB=ON  PARK S?/AU
L48            6 SEA FILE=MEDLINE ABB=ON  L45 AND L46 AND L47
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=> => fil wpix; d que l111
FILE 'WPIX' ENTERED AT 16:05:39 ON 03 MAY 2007
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FILE LAST UPDATED:                          30 APR 2007   <20070430/UP>
MOST RECENT THOMSON SCIENTIFIC UPDATE:     200728       <200728/DW>
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE

>>> New reloaded DWPI Learn File (LWPI) available as well <<<

>>> YOU ARE IN THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX <<<

>>> New display format FRAGHITSTR available <<<
    SEE ONLINE NEWS and
http://www.stn-international.de/archive/stn_online_news/fraghitstr_ex.pdf

>>> IPC Reform backfile reclassification has been loaded to 31 December
    2006. No update date (UP) has been created for the reclassified
    documents, but they can be identified by 20060101/UPIC and
    20061231/UPIC. <<<

FOR A COPY OF THE DERWENT WORLD PATENTS INDEX STN USER GUIDE,
PLEASE VISIT:
http://www.stn-international.de/training_center/patents/stn_guide.pdf

FOR DETAILS OF THE PATENTS COVERED IN CURRENT UPDATES, SEE
http://scientific.thomson.com/support/patents/coverage/latestupdates/

PLEASE BE AWARE OF THE NEW IPC REFORM IN 2006, SEE
http://www.stn-international.de/stndatabases/details/ipc_reform.html and
http://scientific.thomson.com/media/scpdf/ipcrdwpi.pdf
```

```
>>> FOR DETAILS ON THE NEW AND ENHANCED DERWENT WORLD PATENTS INDEX
    PLEASE SEE
http://www.stn-international.de/stndatabases/details/dwpi_r.html <<<
'BI ABEX' IS DEFAULT SEARCH FIELD FOR 'WPIX' FILE


L102         538 SEA FILE=WPIX ABB=ON  JANG I?/AU
L103          16 SEA FILE=WPIX ABB=ON  YEO E?/AU
L104       21388 SEA FILE=WPIX ABB=ON  PARK S?/AU
L105           2 SEA FILE=WPIX ABB=ON  (((TETRAHYDRO/BI,ABEX OR TETRA HYDRO/BI,A
                 BEX)(1W)FURYL/BI,ABEX) OR TETRAHYDROFURYL/BI,ABEX)(1A)ADENINE/B
                 I,ABEX
L106           1 SEA FILE=WPIX ABB=ON  NSC53339/BI,ABEX OR NSC 53339/BI,ABEX OR
                 SQ22536/BI,ABEX OR SQ 22536/BI,ABEX
L111           1 SEA FILE=WPIX ABB=ON  (L102 OR L103 OR L104) AND (L105 OR
                 L106)


=> dup rem l48,l98,l10,l111,l34,l61
FILE 'MEDLINE' ENTERED AT 16:06:01 ON 03 MAY 2007

FILE 'BIOTECHNO' ENTERED AT 16:06:01 ON 03 MAY 2007
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PROCESSING COMPLETED FOR L48
PROCESSING COMPLETED FOR L98
PROCESSING COMPLETED FOR L10
PROCESSING COMPLETED FOR L111
PROCESSING COMPLETED FOR L34
PROCESSING COMPLETED FOR L61
L113          10 DUP REM L48 L98 L10 L111 L34 L61 (16 DUPLICATES REMOVED)
                 ANSWERS '1-6' FROM FILE MEDLINE
                 ANSWER '7' FROM FILE BIOTECHNO
                 ANSWERS '8-9' FROM FILE CAPLUS
                 ANSWER '10' FROM FILE BIOSIS

=> d ibib ed ab 1-10

L113 ANSWER 1 OF 10     MEDLINE on STN                    DUPLICATE 1
ACCESSION NUMBER:    2006187994     MEDLINE  Full-text
DOCUMENT NUMBER:     PubMed ID: 16516270
TITLE:               Lysophosphatidic acid-induced changes in cAMP profiles in
                     young and senescent human fibroblasts as a clue to the
                     ageing process.
AUTHOR:              Jang Ik-Soon; Rhim Ji-Heon; Kim Kyung-Tae; Cho
                     Kyung A; Yeo Eui-Ju; Park Sang Chul
```

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Ageing and Apoptosis Research Center, Seoul National University College of Medicine, Chongno-gu, South Korea.
SOURCE: Mechanisms of ageing and development, (2006 May) Vol. 127, No. 5, pp. 481-9. Electronic Publication: 2006-03-03. Journal code: 0347227. ISSN: 0047-6374.
PUB. COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200606
ENTRY DATE: Entered STN: 5 Apr 2006
Last Updated on STN: 24 Jun 2006
Entered Medline: 23 Jun 2006
ED Entered STN: 5 Apr 2006
Last Updated on STN: 24 Jun 2006
Entered Medline: 23 Jun 2006

AB This study attempts to elucidate the molecular mechanisms underlying the ageing-dependent cAMP profiles in human diploid fibroblasts stimulated by lysophosphatidic acid (LPA). In senescent cells, LPA-dependent Gialpha activation was reduced, with a consequent reduction in Gi-suppressed cAMP levels, without alterations in the levels of Gialpha proteins. In young cells, when Gialpha activity was inhibited by pertussis toxin pretreatment, or when its expression was blocked by siRNA, the pattern of changes in cAMP levels in response to LPA was similar to that seen in senescent cells. An increase in protein kinase C (PKC)-dependent isoforms of adenylyl cyclase (AC) types II, IV, and VI was also observed in these senescent fibroblasts. In senescent cells treated with PKC-specific inhibitors, bis-indolylmaleimide, Go6976, rottlerin, and PKCvarepsilonV1, LPA-induced cAMP accumulation was inhibited, indicating that increased ACs in response to LPA occur via the activation of protein kinase Cs. When the expression of AC II, IV, and VI was blocked by siRNA in senescent fibroblasts, LPA-induced cAMP accumulation was also blocked. These results suggest that the senescence-associated increase of cAMP levels after LPA treatment is associated with reduced Gialpha, increased AC II, IV, and VI proteins, and PKC-dependent stimulation of their activities and provide an explanation for the age-dependent differences in cAMP-related physiological responses.

L113 ANSWER 2 OF 10 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2006694406 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 17081159
TITLE: Role of protein kinase C-dependent A-kinase anchoring proteins in lysophosphatidic acid-induced cAMP signaling in human diploid fibroblasts.
AUTHOR: Rhim Ji-Heon; **Jang Ik-Soon**; **Yeo Eui-Ju**; Song Kye-Yong; **Park Sang Chul**
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Aging and Apoptosis Research Center, Seoul National University College of Medicine, Seoul 110-799, South Korea.
SOURCE: Aging cell, (2006 Dec) Vol. 5, No. 6, pp. 451-61. Electronic Publication: 2006-11-01. Journal code: 101130839. ISSN: 1474-9718.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200701

ENTRY DATE: Entered STN: 30 Nov 2006
Last Updated on STN: 19 Jan 2007
Entered Medline: 18 Jan 2007

ED Entered STN: 30 Nov 2006
Last Updated on STN: 19 Jan 2007
Entered Medline: 18 Jan 2007

AB Previously, we reported that lysophosphatidic acid (LPA)-induced adenosine 3',5'-cyclic monophosphate (cAMP) production by human diploid fibroblasts depends on the age of the fibroblasts. In this study, we examined the role of A-kinase anchoring proteins (AKAP) in the regulation of LPA-stimulated cAMP production in senescent fibroblasts. We found that levels of protein kinase C (PKC)-dependent AKAPs, such as Gravin and AKAP79, were elevated in senescent cells. Co-immunoprecipitation experiments revealed that Gravin and AKAP79 do not associate with adenylyl cyclase type 2 (AC2) but bind to AC4/6, which interacts with calcium-dependent PKCs alpha/beta both in young and senescent fibroblasts. When the expression of Gravin and AKAP79 was blocked by small interference RNA transfection, the basal level of cAMP was greatly reduced and the cAMP status after LPA treatment was also reversed. Protein kinase A showed a similar pattern in terms of its basal activity and LPA-dependent modulation. These data suggest that Gravin and to a lesser extent, AKAP79, may play important roles in maintaining the basal AC activity and in coupling the AC systems to inhibitory signals such as Gialpha in young cells, and to stimulatory signals such as PKCs in senescent cells. This study also demonstrates that Gravin is especially important for the long-term activation of PKC by LPA in senescent cells. We conclude that LPA-dependent increased level of cAMP in senescent human diploid fibroblasts is associated with increases in Gravin levels resulting in its increased binding with and activation of calcium-dependent PKC alpha/beta and AC4/6.

L113 ANSWER 3 OF 10 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2006248476 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16672767
TITLE: Downstream molecular events in the altered profiles of lysophosphatidic acid-induced cAMP in senescent human diploid fibroblasts.
AUTHOR: **Jang Ik Soon**; Rhim Ji Heon; **Park Sang Chul**; **Yeo Eui Ju**
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, Aging and Apoptosis Research Center, Seoul National University College of Medicine, Seoul 110-799, Korea.
SOURCE: Experimental & molecular medicine, (2006 Apr 30) Vol. 38, No. 2, pp. 134-43.
Journal code: 9607880. ISSN: 1226-3613.
PUB. COUNTRY: Korea (South)
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200607
ENTRY DATE: Entered STN: 5 May 2006
Last Updated on STN: 26 Jul 2006
Entered Medline: 25 Jul 2006

ED Entered STN: 5 May 2006
Last Updated on STN: 26 Jul 2006
Entered Medline: 25 Jul 2006

AB Lysophosphatidic acid (LPA) is a phospholipid growth factor that acts through G-protein-coupled receptors. Previously, we demonstrated an altered profile of LPA-dependent cAMP content during the aging process of human diploid fibroblasts (HDFs). In attempts to define the molecular events associated

with the age-dependent changes in cAMP profiles, we determined the protein kinase A (PKA) activity, phosphorylation of cAMP-response element binding protein (CREB), and the protein expression of CRE-regulatory genes, c-fos and COX-2 in young and senescent HDFs. We observed in senescent cells, an increase in mRNA levels of the catalytic subunit a of PKA and of the major regulatory subunit Ialpha. Senescence-associated increase of cAMP after LPA treatment correlated well with increased CREB phosphorylation accompanying activation of PKA in senescent cells. In senescent cells, after LPA treatment, the expression of c-fos and COX-2 decreased initially, followed by an increase. In young HDFs, CREB phosphorylation decreased following LPA treatment, and both c-fos and COX-2 protein levels increased rapidly. CRE-luciferase assay revealed higher basal CRE-dependent gene expression in young HDFs compared to senescent HDFs. However, LPA-dependent slope of luciferase increased more rapidly in senescent cells than in young cells, presumably due to an increase of LPA-induced CREB phosphorylation. CRE-dependent luciferase activation was abrogated in the presence of inhibitors of PKC, MEK1, p38MAPK, and PKA, in both young and senescent HDFs. We conclude that these kinase are coactivators of the expression of CRE-responsive genes in LPA-induced HDFs and that their changed activities during the aging process contribute to the final expression level of CRE-responsive genes.

L113 ANSWER 4 OF 10 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2003132009 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12646237
TITLE: Altered cAMP signaling induced by lysophosphatidic acid in senescent human diploid fibroblasts.
AUTHOR: **Jang Ik-Soon**; **Yeo Eui-Ju**; Park Ji-Ae; Ahn Jeong Soo; Park Jeong Soo; Cho Kyung A; Juhnn Yong-Sung; **Park Sang-Chul**
CORPORATE SOURCE: Department of Biochemistry, Seoul National University College of Medicine, 28 Yon-gon-Dong, Chongno-Gu, 110-799, Seoul, Republic of Korea.
SOURCE: Biochemical and biophysical research communications, (2003 Mar 21) Vol. 302, No. 4, pp. 778-84.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 21 Mar 2003
Last Updated on STN: 14 May 2003
Entered Medline: 13 May 2003

ED Entered STN: 21 Mar 2003
Last Updated on STN: 14 May 2003
Entered Medline: 13 May 2003

AB Lysophosphatidic acid (LPA) is a lipid mitogen that acts through G-protein-coupled receptors. LPA responsiveness has been reported to be dependent on the senescent state of the cells. To solve the mechanism underlying, we observed LPA-dependent cAMP status and found its age-dependent contrasting profile such as high level of cAMP in the senescent cells vs its low level in the young cells. In order to clarify the molecular mechanism of the ageing effect, we examined various molecular species involved in the cAMP signaling pathway by semi-quantitative RT-PCR. EDG-1 and EDG-4 were unchanged, but EDG-2 and EDG-7 were reduced with age. Senescent cells showed a partial reduction of Gi1, Gi2, and Gi3, but no change in the level of Gq. Decreased Gis and Gi-coupled LPA receptors may reduce the inhibitory effect of Gi alpha on adenylyl cyclases (ACs), resulting in cAMP accumulation via activation of adenylyl

cyclase in senescent fibroblasts. We also observed an age-dependent increase in some of AC isoforms: II, IV, and VI. In conclusion, multiple changes in the cAMP signaling pathway of the senescent cells might explain the altered responsiveness to the mitogenic stimuli.

L113 ANSWER 5 OF 10 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2004144994 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 15033777
TITLE: Gelsolin for senescence-associated resistance to apoptosis.
AUTHOR: Ahn Jeong Soo; **Jang Ik-Soon**; Rhim Ji Heon; Kim Kyungtae; **Yeo Eui-Ju**; **Park Sang Chul**
CORPORATE SOURCE: Department of Biochemistry, Seoul National University College of Medicine, Seoul, South Korea.
SOURCE: Annals of the New York Academy of Sciences, (2003 Dec) Vol. 1010, pp. 493-5.
Journal code: 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200406
ENTRY DATE: Entered STN: 25 Mar 2004
Last Updated on STN: 16 Jun 2004
Entered Medline: 15 Jun 2004
ED Entered STN: 25 Mar 2004
Last Updated on STN: 16 Jun 2004
Entered Medline: 15 Jun 2004
AB One of the characteristics of the senescent cell is apoptotic resistance. Gelsolin, a Ca(2+)-dependent actin regulatory protein, is believed to regulate the intracellular movements which are necessary for cell growth, proliferation, and differentiation. Recently, gelsolin was suggested to play a role in apoptotic resistance, which led us to examine its involvement in the apoptotic resistance of senescent cells. We found that the protein and mRNA levels of gelsolin were increased in senescent human diploid fibroblasts (HDFs). Gelsolin was intracellularly co-localized to the actin stress fiber and distributed to the nucleus and mitochondria in old HDFs. To examine the anti-apoptotic function of gelsolin in senescent HDFs, we tried to downregulate the expression of gelsolin by using antisense oligonucleotide in old HDFs. We then treated the senescent HDFs with the apoptosis-inducing agent menadione. Downregulation of gelsolin in senescent HDFs resulted in increased sensitivity to menadione-induced apoptotic cell death. This suggests that gelsolin plays a role in the apoptotic resistance observed in senescent HDFs.

L113 ANSWER 6 OF 10 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2002344254 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 12086695
TITLE: Agonist-specific differential changes of cellular signal transduction pathways in senescent human diploid fibroblasts.
AUTHOR: **Yeo Eui-Ju**; **Jang Ik-Soon**; Lim Hee-Kyoung; Ha Kwon-Soo; **Park Sang Chul**
CORPORATE SOURCE: Department of Biochemistry, Gachon Medical School, Inchon 417-840, South Korea.
SOURCE: Experimental gerontology, (2002 Jul) Vol. 37, No. 7, pp. 871-83.
Journal code: 0047061. ISSN: 0531-5565.
PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 28 Jun 2002
Last Updated on STN: 27 Sep 2002
Entered Medline: 26 Sep 2002
ED Entered STN: 28 Jun 2002
Last Updated on STN: 27 Sep 2002
Entered Medline: 26 Sep 2002

AB Changes in the signal transduction efficiency of senescent cells led us to compare the signaling events induced by two mitogenic agonists, platelet-derived growth factor (PDGF) and lysophosphatidic acid (LPA) in presenescent and senescent or near-senescent human diploid fibroblasts. When the changes in intracellular [Ca(2+)](i) were analyzed, both PDGF and LPA generated a rhythmic increase in [Ca(2+)](i) in presenescent cells. The frequency of calcium response was reduced and desensitized in PDGF-stimulated senescent cells, while response to a LPA-induced calcium signal was also reduced in frequency, though its magnitude was unaltered. PDGF treatment increased the fibrous actin (F-actin) level in presenescent cells but not in senescent cells in contrast to a reduced but visible increase in F-actin in LPA-treated senescent cells. The effect of PDGF on phospholipase D (PLD) activation was also reduced significantly, as a ca. 60-80% reduction of PLD activity was observed in PDGF-stimulated cells but only a little reduction in LPA-induced cells. Agonist-specific differential changes of cellular signaling events caused a differential effect on DNA synthesis after growth factor stimulation. We observed a dramatic (80-90%) reduction of [3H]thymidine incorporation into DNA in the PDGF-stimulated near-senescent cells. LPA resulted in a 2-3-fold increase in thymidine incorporation even in the near-senescent cells. These differences in the responses of senescent or near-senescent cells to PDGF- and LPA-stimulation raised questions about the differential changes of the respective signaling apparatuses induced by aging. Since PDGF signaling event was affected greatly by aging, we further examined the protein contents involved in PDGF signal transduction pathway. PDGF receptor (PDGFR), protein kinase C-alpha (PKC-alpha), phospholipase C-gamma1 (PLC-gamma1), and PLD1 were examined by Western blot analysis. The protein levels of PKC-alpha and PLC-gamma1 were unchanged, but those of PLD1 and PDGFR were reduced with age. The reduced content of PDGFR protein may be one of the important contributors to the failure of PDGF-stimulated signal transduction in human senescent fibroblasts. Our results strongly suggest that age-dependent agonist-specific changes in signaling events might be in charge of the functional deterioration of senescent cells through imbalance of signal responses.

L113 ANSWER 7 OF 10 BIOTECHNO COPYRIGHT 2007 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2001:32222421 BIOTECHNO Full-text
TITLE: Regulation of phosphate uptake in primary cultured rabbit renal proximal tubule cells by glucocorticoids: Evidence for nongenomic as well as genomic mechanisms
AUTHOR: **Park S.-H.**; Taub M.; Han H.-J.
CORPORATE SOURCE: H.-J. Han, Department of Veterinary Physiology, College of Veterinary Medicine, Chonnam National University, Kwangju 500-757, South Korea.
E-mail: hjhan@chonnam.chonnam.ac.kr
SOURCE: Endocrinology, (2001), 142/2 (710-720), 54 reference(s)
CODEN: ENDOAO ISSN: 0013-7227
DOCUMENT TYPE: Journal; Article
COUNTRY: United States

LANGUAGE: English
SUMMARY LANGUAGE: English
ED 20010404
AB We have investigated the nongenomic as well as the genomic effects of glucocorticoids on phosphate (Pi) uptake in primary rabbit renal proximal tubule cells (PTCs) and have defined the involved signaling pathways. In the present study, cortisol-BSA (cortisol-BSA) (> 10.sup.-.sup.9 M, 30 rain) was found to inhibit Pi uptake in a time- and concentration- dependent manner. However, progesterone-BSA (P.sub.4-BSA), 17βestradiol-BSA (E.sub.2-BSA), testosterone-BSA (T.sub.4-BSA), aldosterone, P.sub.4, E.sub.2, and T.sub.4 (10.sup.-.sup.9 M, 1 h) had no effect on Pi uptake. In addition, cortisol-BSA (10.sup.-.sup.9 M) did not affect either Na.sup.+ uptake or α-methylglucopyranoside (α-MG) uptake. The cortisol-BSA-induced inhibition of Pi uptake was associated with a decrease in the V.sub.m.sub.a.sub.x for Pi uptake, rather than the K.sub.m. The inhibitory effect of cortisol-BSA was not blocked either by actinomycin D (an inhibitor of transcription), cycloheximide (an inhibitor of translation), or classical glucocorticoid receptor antagonists (RU 486 or P.sub.4). The cortisol-BSA-induced inhibition of Pi uptake was blocked by two phospholipase C (PLC) inhibitors (neomycin or U73122), and two protein kinase C (PKC) inhibitors (staurosporine or bisindolylmaleimide I) but not by two adenylate cyclase/protein kinase A inhibitors [**SQ 22536** (an adenylate cyclase inhibitor) or myristoylated protein kinase A inhibitor amide 14-22]. Furthermore, cortisol-BSA promoted the translocation of PKC from the cytosolic fraction to the membrane fraction, while having no effect on the activity of adenylate cyclase. Our observations may thus be interpreted as indicating that cortisol does indeed inhibit renal Pi uptake via a nongenomic mechanism, which involves the PLC/PKC pathway.

L113 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2003:991699 CAPLUS Full-text
DOCUMENT NUMBER: 140:39513
TITLE: Signals and molecular species involved in senescence, detection of senescent cells and compositions for modulating cellular senescence
INVENTOR(S): **Jang, Ik-soon; Yeo, Eui-ju; Park, Sang-chul**
PATENT ASSIGNEE(S): Metabolic Engineering Laboratories Co., Ltd., S. Korea
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003104482	A1	20031218	WO 2002-KR1067	20020605
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				

AU 2002303023 A1 20031222 AU 2002-303023 20020605
US 2006099568 A1 20060511 US 2005-517269 20050926 <--
PRIORITY APPLN. INFO.: WO 2002-KR1067 A 20020605
ED Entered STN: 21 Dec 2003
AB The present invention relates to (a) a method for detecting a human senescent cell, which comprises measuring a relative alteration to young cell in a signal or mol. species involved in signal transduction triggered by platelet-derived growth factor or lysophosphatidic acid; (b) a method and a composition for modulating cellular senescence comprising treating a senescent cell with the effective amount of an inhibitor of adenylyl cyclase or an inhibitor of protein kinase A. The alteration in signal or mol. species is selected from the group consisting of: (a) a reduction in Ca2+ oscillation; (b) a reduction in expression of F-actin; (c) a reduction in activity of phospholipase C; (d) a reduction in activity of phospholipase D; (e) a reduction in expression or phosphorylation of platelet-derived growth factor receptor; (f) a reduction in phosphorylation of phospholipase C-γ1; (g) a reduction in expression of phospholipase D1; (h) a reduction in expression of EDG (endothelial differentiation gene)-2; (i) a reduction in expression of EDG-7; (j) a reduction in expression of Gi1; (k) a reduction in expression of Gi2; (l) a reduction in expression of Gi3; (m) an increase in activity or expression of adenylyl cyclase; (n) a reduction in activity or expression of phosphodiesterase; (o) an increase in activity of protein kinase C; (p) an increase in activity or expression of protein kinase A; (q) an increase in phosphorylation of CREB; and (r) an increase in cAMP content.
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1999:413706 CAPLUS Full-text
DOCUMENT NUMBER: 131:68439
TITLE: Leukotriene D4 inhibits Na+ uptake through cAMP and PLC pathways in primary cultured renal proximal tubular cells
AUTHOR(S): Han, Ho-Jae; **Park, Soo-Hyun**; Lee, Jae-Cheon; Lee, Hwanghee-Blasie; Park, Haeng-Soon
CORPORATE SOURCE: Dep. Veterinary Physiology, College Veterinary Medicine, Chonnam National Univ., Kwangju, 500757, S. Korea
SOURCE: Kidney & Blood Pressure Research (1999), 22(3), 106-113
CODEN: KBPRFC; ISSN: 1420-4096
PUBLISHER: S. Karger AG
DOCUMENT TYPE: Journal
LANGUAGE: English
ED Entered STN: 05 Jul 1999
AB The effect was investigated of leukotriene D4 (LTD4) on Na+ uptake and its related signal transduction pathways in renal proximal tubular cells (PTCs). LTD4 (>10-9 M) inhibited the Na+ uptake after 15 min (controls vs. LTD4 10-9 M: 431.7 vs. 355.0 nmol/mg protein) and its effect was blocked by MK-571 (10-6 M), a leukotriene receptor antagonist, in PTCs. Preincubation with cilastatin, a renal dipeptidase inhibitor, and polyclonal antibody against renal dipeptidase potentiated the inhibitory effect of LTD4 on Na+ uptake. SQ 22536 (10-6 M), an adenylate cyclase inhibitor, and the myristoylated protein kinase A inhibitor amide 14-22 (PKI, 10-5 M) blocked the effect of LTD4 on Na+ uptake (LTD4 vs. SQ 22536+LTD4 and PKI+LTD4: 349.9 vs. 476.5 and 440.3 nmol/mg protein), and LTD4 induced an increase in cAMP, suggesting the involvement of cAMP in the inhibition of Na+ uptake. Addnl., U 73122 (10-6M) and neomycin (10-4 M), phospholipase C (PLC) inhibitors, W-7 (10-4 M), a calmodulin antagonist, and bisindolylmaleimide I, a protein kinase C (PKC) inhibitor, blocked the LTD4-induced inhibition of Na+ uptake, strongly suggesting

involvement of the PLC-PKC signal pathways in the effect of LTD4. LTD4 increased [Ca2]i by 49% as compared with baseline. TMB-8 (10-5 M) and BAPTA/AM (10-5M), intracellular Ca mobilization blockers, completely blocked the LTD4-induced inhibition of Na+ uptake (LTD4 vs. TMB-8+LTD4 and BAPTA/AM+LTD4: 347.6 vs. 436.4 and 419.9 nmol/mg protein). EGTA (1 mM), a Ca chelator, partially blocked the LTD4-induced inhibition of Na+ uptake. LTD4-induced inhibition of Na+ uptake was supposed to be involved in both cAMP and PLC-PKC signal pathways in PTCs. Ca2+ coming from the intracellular Ca2+ mobilization was primarily responsible for the LTD4-induced inhibition of Na+ uptake.

REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L113 ANSWER 10 OF 10 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:230646 BIOSIS Full-text
DOCUMENT NUMBER: PREV200400231025
TITLE: Gelsolin for senescence-associated resistance to apoptosis.
AUTHOR(S): Ahn, Jeong Soo; **Jang, Ik-Soon**; Rhim, Ji Heon; Kim, Kyungtae; **Yeo, Eui-Ju**; **Park, Sang Chul** [Reprint Author]
CORPORATE SOURCE: Department of Biochemistry, College of Medicine, Seoul National University, 28 Yon-gon-Dong, Chongno-Gu, Seoul, 110-799, South Korea
scpark@snu.ac.kr
SOURCE: Diederich, Marc [Editor, Reprint Author]. (2003) pp. 493-495. Apoptosis: From signaling pathways to therapeutic tools. print.
Publisher: New York Academy of Sciences, 2 East 63rd Street, New York, NY, 10021, USA. Series: Annals of the New York Academy of Sciences.
ISSN: 0077-8923 (ISSN print). ISBN: 1-57331-474-9 (cloth), 1-57331-475-7 (paper).
DOCUMENT TYPE: Book; (Book Chapter)
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Apr 2004
Last Updated on STN: 28 Apr 2004
ED Entered STN: 28 Apr 2004
Last Updated on STN: 28 Apr 2004

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TEXT SEARCH
=>
=> => fil biosis;d que l40; s l40 not l34
FILE 'BIOSIS' ENTERED AT 16:07:47 ON 03 MAY 2007
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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 2 May 2007 (20070502/ED)


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                 URANYL)-"/CN OR "9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-FURANYL)-,
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L22          235 SEA FILE=BIOSIS ABB=ON  L3
L26        23855 SEA FILE=BIOSIS ABB=ON  SENESCEN?
L30          356 SEA FILE=BIOSIS ABB=ON  NSC53339 OR NSC 53339 OR SQ22536 OR SQ
                 22536
L31           60 SEA FILE=BIOSIS ABB=ON  (((TETRAHYDRO OR TETRA HYDRO)(1W)FURYL)
                  OR TETRAHYDROFURYL)(1A)ADENINE
L39       110377 SEA FILE=BIOSIS ABB=ON  AGING OR AGEING
L40            1 SEA FILE=BIOSIS ABB=ON  (L22 OR L30 OR L31) AND (L26 OR L39)


L114           1 L40 NOT L34

=> fil biotechno; d que l101; s l101 not l98

FILE 'BIOTECHNO' ENTERED AT 16:07:48 ON 03 MAY 2007
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FILE LAST UPDATED: 7 JAN 2004       <20040107/UP>
FILE COVERS 1980 TO 2003.

>>>  BIOTECHNO IS NO LONGER BEING UPDATED AS OF 2004  <<<

>>>  SIMULTANEOUS LEFT AND RIGHT TRUNCATION AVAILABLE IN
     /CT AND BASIC INDEX  <<<


L3             2 SEA FILE=REGISTRY ABB=ON  ("9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-F
                 URANYL)-"/CN OR "9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-FURANYL)-,
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L90           59 SEA FILE=BIOTECHNO ABB=ON  JANG I?/AU
L91           22 SEA FILE=BIOTECHNO ABB=ON  YEO E?/AU
L92         1417 SEA FILE=BIOTECHNO ABB=ON  PARK S?/AU
L93           78 SEA FILE=BIOTECHNO ABB=ON  L3
L94           84 SEA FILE=BIOTECHNO ABB=ON  (((TETRAHYDRO OR TETRA HYDRO)(1W)FUR
                 YL) OR TETRAHYDROFURYL)(1A)ADENINE
L95         3797 SEA FILE=BIOTECHNO ABB=ON  SENESCEN?
L96         9713 SEA FILE=BIOTECHNO ABB=ON  AGING OR AGEING
L97           75 SEA FILE=BIOTECHNO ABB=ON  NSC53339 OR NSC 53339 OR SQ22536 OR
                 SQ 22536
L98            2 SEA FILE=BIOTECHNO ABB=ON  (L90 AND L91 AND L92) OR ((L90 OR
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L101          1 SEA FILE=BIOTECHNO ABB=ON  (L93 OR L94 OR L98) AND (L95 OR
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L115          0 L101 NOT L98


=> fil drugu; d que l89

FILE 'DRUGU' ENTERED AT 16:07:51 ON 03 MAY 2007
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FILE LAST UPDATED: 3 MAY 2007        <20070503/UP>
>>>  DERWENT DRUG FILE (SUBSCRIBER)  <<<

>>>  FILE COVERS 1983 TO DATE  <<<
>>>  THESAURUS AVAILABLE IN /CT  <<<



L3            2 SEA FILE=REGISTRY ABB=ON  ("9H-PURIN-6-AMINE, 9-(TETRAHYDRO-2-F
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L80          76 SEA FILE=DRUGU ABB=ON  SQ-22536/CT
L81        2140 SEA FILE=DRUGU ABB=ON  ADENYLATE-CYCLASE/CT
L85         566 SEA FILE=DRUGU ABB=ON  SENESCEN?
L86        3074 SEA FILE=DRUGU ABB=ON  AGING OR AGEING
L87           0 SEA FILE=DRUGU ABB=ON  (L79 OR L80) AND (L85 OR L86)
L88           8 SEA FILE=DRUGU ABB=ON  (L79 OR L80) AND L81
L89           8 SEA FILE=DRUGU ABB=ON  (L87 OR L88)


=> fil embase; d que l75; s l75 not l61

FILE 'EMBASE' ENTERED AT 16:07:52 ON 03 MAY 2007
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 FILE COVERS 1974 TO 3 May 2007 (20070503/ED)

 EMBASE is now updated daily.  SDI frequency remains weekly (default)
 and biweekly.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


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L57         428 SEA FILE=EMBASE ABB=ON  "9 (TETRAHYDRO 2 FURYL)ADENINE"/CT
L58       21524 SEA FILE=EMBASE ABB=ON  ADENYLATE CYCLASE/CT
L60      182487 SEA FILE=EMBASE ABB=ON  "CELL AGING, CELL DEGENERATION AND
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L72        8124 SEA FILE=EMBASE ABB=ON  SENESCENCE/CT
L73       76576 SEA FILE=EMBASE ABB=ON  AGING/CT
L74           9 SEA FILE=EMBASE ABB=ON  (L56 OR L57) AND (L60 OR L72 OR L73)
L75           9 SEA FILE=EMBASE ABB=ON  L74 OR (L74 AND L58)
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